Cardiovascular disease is the leading cause of morbidity and mortality in patients with chronic kidney disease (CKD).\[1,2\] CKD is associated with increased prevalence of concomitant congestive heart failure, coronary heart disease (CHD), cardiac arrhythmias, valvular calcification, cerebrovascular disease, stroke, and peripheral vascular disease.\[3\] Increased risk of cardiovascular events in these patients is related to traditional and nontraditional risk factors.\[4–6\] Traditional risk factors such as diabetes mellitus, hypertension, and dyslipidemia are more prevalent in patients with CKD than in the general population.\[5–7\] Moreover, nontraditional risk factors for cardiovascular disease, including albuminuria, anemia, hyperphosphatemia, oxidative stress, and inflammation, are common in patients with CKD.\[6\] Therefore, CKD is accepted as a risk factor for the development and progression of cardiovascular disease.\[8\] The American Heart Association advises that all patients with cardiovascular disease be screened for evidence of kidney disease by calculating glomerular filtration rate (GFR) and detecting microalbuminuria.\[9\]

CKD is associated with accelerated cardiovascular disease, even when kidney function is only mildly decreased. Several studies have shown that mild-to-moderate elevations in serum creatinine levels independently increase risk of cardiovascular morbidity and mortality.\[9–11\] Go et al.\[9\] reported that among 1,120,295 adults within a large, integrated system of health care delivery, a reduced estimated GFR was associated with increased risk of death, cardiovascular events, and hospitalization, independent of known risk factors, history of cardiovascular disease, and presence of documented proteinuria. Likewise, Anavekar et al.\[11\] found that even a mild decrease in GFR is an independent and easily identifiable risk factor for cardiovascular complications among patients who have had myocardial infarction. Furthermore, patients with CKD have higher risk of morbidity and mortality following coronary revascularization procedures, compared to patients with normal renal function.\[12–14\]

In this issue of the Archives of the Turkish Society of Cardiology, Ekici et al.\[15\] investigated the effects of GFR on the severity of CHD. The authors included 918 patients who had undergone coronary angiography due to positive noninvasive stress test. The extent and severity of CHD were evaluated according to SYNTAX score, and GFR was calculated using the simplified modification of diet in renal disease equation. A negative, statistically significant correlation was determined between SYNTAX score and GFR.

**Abbreviations:**

- **CHD** Coronary heart disease
- **CKD** Chronic kidney disease
- **GFR** Glomerular filtration rate
In a previous study that included 411 patients with stable coronary artery disease, Uçar et al.[16] similarly reported that patients with GFR <90 ml/min/1.73 m² had significantly higher SYNTAX scores than patients with GFR ≥90 ml/min/1.73 m². The Ekici et al.[15] study, which included a larger patient population, confirmed this finding. Moreover, a novel finding in this study is that patients with lower GFRs were more likely to undergo percutaneous coronary intervention and coronary artery bypass graft, compared to patients with higher GFRs, who were more likely to continue with medical therapy.

In conclusion, there is growing evidence in epidemiological and clinical studies that decreased GFR is an independent risk factor for cardiovascular morbidity and mortality, both in patients with CKD and in patients with CHD. GFR can be easily calculated in order to predict patient risk. Can GFR be used as a marker of severity of coronary artery disease? The study by Ekici et al.[15] provides this evidence. More studies may support this approach in the future.

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